Exp Brain Res (2014) 232:2891–2898
DOI 10.1007/s00221-014-3971-4

RESEARCH ARTICLE

# Afternystagmus in darkness after suppression of optokinetic nystagmus: an interaction of motion aftereffect and retinal afterimages

**Chien-Cheng Chen · Melody Ying-Yu Huang · Konrad P. Weber · Dominik Straumann · Christopher J. Bockisch**

Received: 10 December 2013 / Accepted: 19 April 2014 / Published online: 13 May 2014


**Abstract** The afternystagmus that occurs in the dark after gaze fixation during optokinetic stimulation is directed in the opposite direction relative to the previous optokinetic stimulus. The mechanism responsible for such afternystagmus after suppression of optokinetic nystagmus (ASOKN) is unclear. Several hypotheses have been put forward to explain it, but none is conclusive. We hypothesized that ASOKN is driven by the interaction of two mechanisms: (1) motion-aftereffect (MAE)-induced eye movements and (2) retinal afterimages (RAIs) produced by fixation during the suppression of optokinetic nystagmus (OKN). We examined the correlation among ASOKN, MAE-induced eye movements, and RAIs in healthy subjects. Adapting stimuli consisted of moving random dot patterns and a fixation spot and their brightness was adjusted to induce different RAI durations. Test patterns were a stationary random dot pattern (to test for the presence of a MAE), a dim homogeneous background (to test for MAE driven eye movements), and a black background (to test for ASOKN and RAIs). MAEs were reported by 16 out of 17 subjects, but only 7 out of 17 subjects demonstrated MAE-induced eye movements. Importantly, ASOKN was only found when these seven subjects reported a RAI after suppression of OKN. Moreover, the duration of ASOKN was longer for high-brightness stimuli compared with low-brightness stimuli, just as RAIs persist longer with increasing brightness. We conclude that ASOKN results from the interaction of MAE-induced eye movements and RAIs.

**Keywords** Afternystagmus after suppression of optokinetic nystagmus (ASOKN) · Motion aftereffect (MAE) · Motion-aftereffect-induced eye movements · Retinal afterimage (RAI) · Human

C.-C. Chen · M. Y.-Y. Huang · K. P. Weber · D. Straumann · C. J. Bockisch (✉)
Department of Neurology, University Hospital Zurich, Frauenklinikstrasse 26, 8091 Zurich, Switzerland
e-mail: Chris.Bockisch@usz.ch

C.-C. Chen
PhD Program in Integrative Molecular Medicine, Life Science Graduate School, Zurich, Switzerland

M. Y.-Y. Huang · D. Straumann
Zurich Center for Integrative Human Physiology (ZIHP), Zurich, Switzerland

K. P. Weber · C. J. Bockisch
Department of Ophthalmology, University Hospital Zurich, Zurich, Switzerland

C. J. Bockisch
Department of ENT, University Hospital Zurich, Zurich, Switzerland

## Introduction

Optokinetic nystagmus (OKN), which is induced by a constantly moving large-field visual surround, consists of slow-phase eye movements to minimize retinal slip velocity and fast-phase eye movements for position reset. When the optokinetic stimulus is replaced by total darkness, nystagmus continues with a gradual decay (Ohm 1921). Optokinetic afternystagmus (OKAN) is thought to be generated by a mechanism that stores slow-phase eye velocity signals during optokinetic stimulation and keeps releasing the eye velocity signal for a certain period after cessation of the optokinetic stimulus. This so-called velocity storage mechanism (VSM) stores velocity signals from the optokinetic and vestibular systems to preserve their low frequencies and hence enhance visual–vestibular cooperation during

rotation in the light (Cohen et al. 1977; Raphan et al. 1977, 1979; Laurens and Angelaki 2011).

A different afternystagmus in total darkness occurs after optokinetic stimulation, during which subjects suppress OKN by fixating their eyes upon a space-fixed visual target. This afternystagmus after suppression of OKN (in the following abbreviated with ASOKN) is directed opposite of the previous optokinetic stimulus (Korenaga et al. 1996), which is in contrast to OKAN, during which the nystagmus occurs in the same direction. Kudo et al. (2002) suggested that this "reverse OKAN" results from the VSM, which is charged by retinal slip (a sensory signal) and then generates an eye velocity signal in the opposite direction of optokinetic stimulation to counteract the optokinetic signal. This keeps the fovea on a stationary target in the presence of the moving large-field visual surround. In a different interpretation, Ventre-Dominey and Luyat (2009) suggested that ASOKN is linked to the vestibular and pursuit motor systems rather than a purely sensory signal (retinal slip). Note, however, that ASOKN has not always been found; e.g. Fletcher et al. (1990) found that fixation of a small target during optokinetic stimulation almost completely prevented the development of afternystagmus and thus concluded that gaze fixation actively prevents storage of visual signals.

Gaze fixation upon a space-fixed target during optokinetic stimulation can induce a perceptual motion aftereffect (MAE). The perceptual MAE is an illusory motion perception: A stationary stimulus is perceived to move in the opposite direction of the previous optokinetic stimulus (Anstis et al. 1998; Mather et al. 1998). The perceptual MAE may drive eyes to pursue the illusory motion. Watamaniuk and Heinen (2007) demonstrated that after gaze fixation during unidirectional optokinetic stimulation, eye movements were biased in the opposite direction. In the same study, these authors showed that such MAE-induced eye movements do not occur in the dark, i.e. only appear in the presence of a visual input (they used a moving random dot stimulus). This is analogous to the perceptual MAE, which also requires a visual stimulus (Wohlgemuth 1911; Spigel 1960; Verstraten et al. 1994; Thompson and Wright 1994).

Prolonged ocular fixation of a visual target may induce retinal afterimages (RAIs) (Brown 1965). RAIs, which occur on the retina due to adaptation of photoreceptors, are optical illusions that persist after the exposure to the original image (Shimojo et al. 2001). It has been demonstrated that the duration of RAIs is positively correlated with the intensity and lasting time of the original image (Granit et al. 1930; Feinbloom 1938; Nagamata 1951; Lu et al. 2006). Thus, during optokinetic stimulation, ocular fixation of a space-fixed visual target may induce a RAI as well.

We hypothesized that ASOKN is caused by an interaction of MAE-induced eye movements and RAIs and not the result of any VSM. Although MAE-induced eye movements depend on visual input and should not be activated in the dark, RAIs can be an alternative visual source to elicit these eye movements. Consequently, such eye movements would be directed opposite of the previous optokinetic stimulus. Moreover, since RAIs are eye-fixed, they would move with the MAE-induced movements of the eyes, thereby stimulating further eye movements. Thus, the interaction of MAE-induced eye movements and RAIs could form a positive feedback loop to keep the eyes moving, i.e. nystagmus, in the direction of the MAE-induced eye movements as long as RAIs are present. Such a mechanism can maintain nystagmus independent of the VSM.

To test our hypothesis, we did a correlation study among MAE-induced eye movements, RAIs, and ASOKN in healthy human subjects. Moreover, visual stimuli of different brightness were used to evoke different RAI durations. According to our hypothesis, ASOKN would only occur in those subjects who demonstrate MAE-induced eye movements and perceive a RAI. In addition, the duration of ASOKN should be longer after higher brightness of the stimulus, just as RAIs persist longer with increasing brightness.

## Materials and methods

### Human subjects

Experiments were performed on 10 males and 7 females, aged 23–49 years, with no abnormal neurologic or ophthalmologic history. Visual acuity was normal or corrected to normal. All subjects gave their informed written consent, and the experiment was approved by the local ethics committee.

### Experimental setup

A head-mounted video-oculography (VOG) device (EyeSeeCam, Munich, German) running at a frame rate of 220 Hz was used for the eye-movement recording. Subjects faced the centre of a screen which was placed 100 cm away from the head of subject. The screen was 178 cm in width and 130 cm in height, which hence covered 80° of the horizontal visual field and 66° of the vertical visual field. A digital projector (Panasonic PT-AE7000 Projector), operating at 60 frames per second and a spatial resolution of 1,920 × 1,080 pixels, was used. A custom-developed script in MATLAB (MathWorks, Natick, MA, USA) and its Psychophysics Toolbox extensions (Brainard 1997; Pelli 1997; Kleiner et al. 2007) were applied to control visual stimuli.

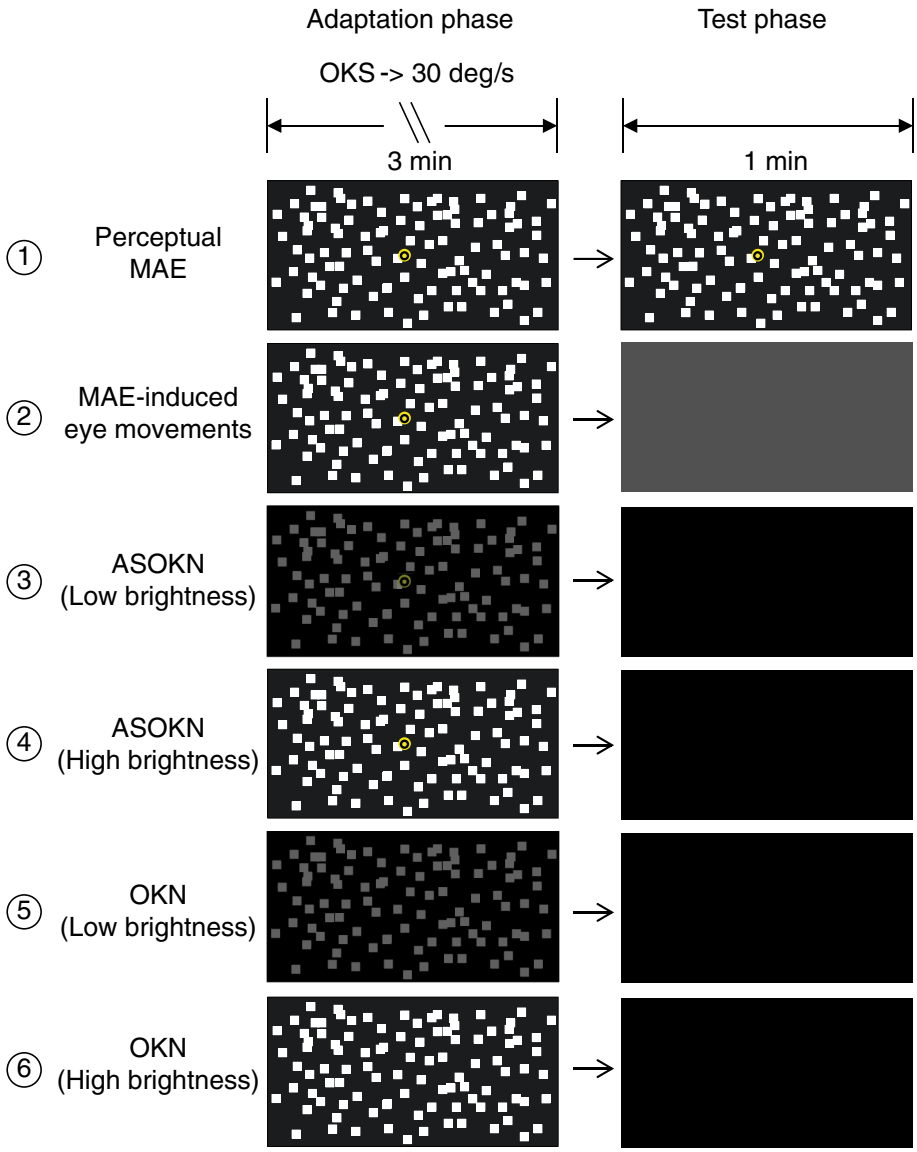


**Fig. 1** Experimental conditions. *Each row* represents the stimuli in a condition. The *left column* shows the motion stimuli (of different brightness), with or without a central fixation dot during the first adaptation phase of each condition. The *right column* represents the stimuli in the testing phase. Condition 1 was applied to test the perceptual MAE. *Dots* of the random dot pattern in the testing phase did not move. Condition 2 was applied to test the MAE-induced eye movements. Conditions 3 and 4 were designed to investigate how stimulus brightness affects ASOKN. Conditions 5 and 6, in which there was no central fixation spot, were designed to investigate how stimulus brightness affects the optokinetic response. The high-brightness visual stimuli were applied on Conditions 1, 2, 4, and 6 while the low brightness ones were applied on Conditions 3 and 5 (see the "Visual stimulation" section of Methods for the details of the stimulus brightness)

Eye position was calibrated at ±10° of the centre of the screen with a custom-developed script.

## Visual stimulation

The optokinetic stimulus was a random pattern of 600 white dots (3 $deg^2$) with a dot lifetime of 150 ms. The speed of each dot was 30 deg/s towards the right. In conditions with gaze fixation, a space-fixed 1.6 $deg^2$ yellow dot with a black ring inside was projected on the centre of the screen (Fig. 1). Low- and high-brightness conditions were used to evoke different RAI durations. In the high-brightness condition, the luminance of the moving dots and the yellow fixation dot was 330 and 314 lux, respectively. In the low-brightness condition, the luminance of the moving dots and the yellow fixation dot was 68 and 70 lux, respectively.

## Experimental conditions

Six conditions were applied in this study (Fig. 1). The sequence of conditions was randomized in each subject. Each condition was presented to subjects once.

Conditions 1–4 started with a 3-min period of suppression of OKN and were followed by a 1-min task. During the period of suppression of OKN, subjects were asked to fixate their eyes upon the yellow stationary dot in the centre of the screen.

Condition 1 tested whether or not the perceptual MAE occurred after suppression of OKN. After suppression of OKN, the speed of each random dot was set to zero during the 1-min task. In other words, each random dot just stayed in the same position for 150 ms and then reappeared in another position. The central yellow fixation dot remained unchanged. Subjects were asked to report any percept of

motion in the random dot pattern while fixating the central yellow dot. The stimuli used in Condition 1 were of high brightness.

With Condition 2, we tested whether or not MAE-induced eye movements occurred. The period of suppression of OKN was the same as in Condition 1. After suppression of OKN, no structured pattern was present on the screen. Thus, the screen luminance (0.17 lux) was uniform across the whole screen. Such a visual environment provides a visual input without fixation targets to evoke possible MAE-induced eye movements.

Conditions 3 (low brightness) and 4 (high brightness) were applied to test how afternystagmus after suppression of OKN (ASOKN), if present, changed with the RAI duration. After suppression of OKN, the visual surround was switched to dark. The dark condition was always examined before Conditions 3 and 4 by asking subjects whether they saw any light while they were covered by a lightproof cloth. Since the light from the LCD beamer we used could not be turned completely off, we found that using the cloth to completely cover the subjects head was a fast and effective way to create a dark environment for the subject. Afterward, subjects were asked whether they saw a RAI during the last minute.

Conditions 5 and 6 were applied to test whether or not low- and high-brightness optokinetic stimuli could evoke the same degree of optokinetic response (OKR). No fixation was provided in these two conditions. The low-brightness condition was used in Condition 5 and the high-brightness condition was used in Condition 6.

### Data analysis

The movement of the left eye was analysed. Data analysis was all done by a custom-developed program written in MATLAB. Eye position was smoothened by a Gaussian low-pass filter with a cut-off frequency of 18 Hz. Eye velocity was derived from the smoothed position signal. Blinks were deleted by visual inspection. Fast-phase selection was done by (1) dividing the whole eye position curve into segments based on the eye-movement direction and (2) identifying a segment as a saccade if its maximum velocity was >30 deg/s. The other segments with a maximum velocity <30 deg/s would be identified as part of slow phase. The duration of ASOKN and MAE-induced eye movements was determined by the number and direction of fast phase. Nystagmus was identified by the fast phases continuously moving towards one direction more than five times. The duration was counted from the first fast phase to the last one. For instance, eye movements shown in Fig. 2a, Condition 2, would be recognized as nystagmus because the eye movements consisted of eight fast phases moving uninterruptedly in one direction and the duration from the first fast phase to the last one was approximately 9 s. Maximum slow-phase eye velocities were calculated by searching for the maximum eye velocity in the opposite direction of the fast phases. Statistical tests (paired one-tailed $t$ test) were done in MATLAB with the Statistics Toolbox.

## Results

In Conditions 1–4, all subjects were able to maintain gaze fixation while a random dot pattern was moving in the background (see Fig. 2). In all subjects, the absolute median eye velocity was <1 deg/s during the period of suppression of OKN. Conditions 5 (low brightness) and 6 (high brightness) tested whether or not low- and high-brightness optokinetic stimuli would evoke similar slow-phase eye velocity during steady-state OKN. The average median eye velocity in Condition 5 (low brightness) was 17.6 ± 8.1 deg/s while the same value in Condition 6 (high brightness) was 19.0 ± 7.4 deg/s; and the difference was not significant (paired one-tailed $t$ test, $t = 1.61$, $p = 0.1272$).

### Perceptual motion aftereffect

After suppression of OKN, 16 out of 17 subjects reported that the space-fixed random dot pattern, in which each random dot stayed in the same position for 150 ms and then reappeared in other positions, was perceived to move towards the left (Table 1). Since all subjects were asked to stare at the yellow fixed dot after suppression of OKN, gaze remained stable (see Fig. 2ab, Condition 1 for examples).

### Motion-aftereffect-induced eye movements

Whether or not MAE-induced eye movements appeared in subjects was evaluated in Condition 2. After suppression of OKN, 7 out of 17 subjects generated a nystagmus in the opposite direction of the previous optokinetic stimulation as they faced the stationary screen with uniformly distributed dim light (example: Fig. 2a, Condition 2), while no nystagmus was recorded in the others (example: Fig. 2b, Condition 2).

### Retinal afterimage (RAI)

Subjects were asked whether RAIs appeared after suppression of OKN in Conditions 3 (low brightness) and 4 (high brightness). Eleven subjects reported that a RAI appeared after the low-brightness condition (Condition 3) while 15 subjects saw a RAI after the high-brightness condition (Condition 4). Ten of 11 subjects who saw a RAI in both conditions reported that the RAI after the high-brightness condition lasted longer than after the low-brightness condition, while 1 subject reported no difference.

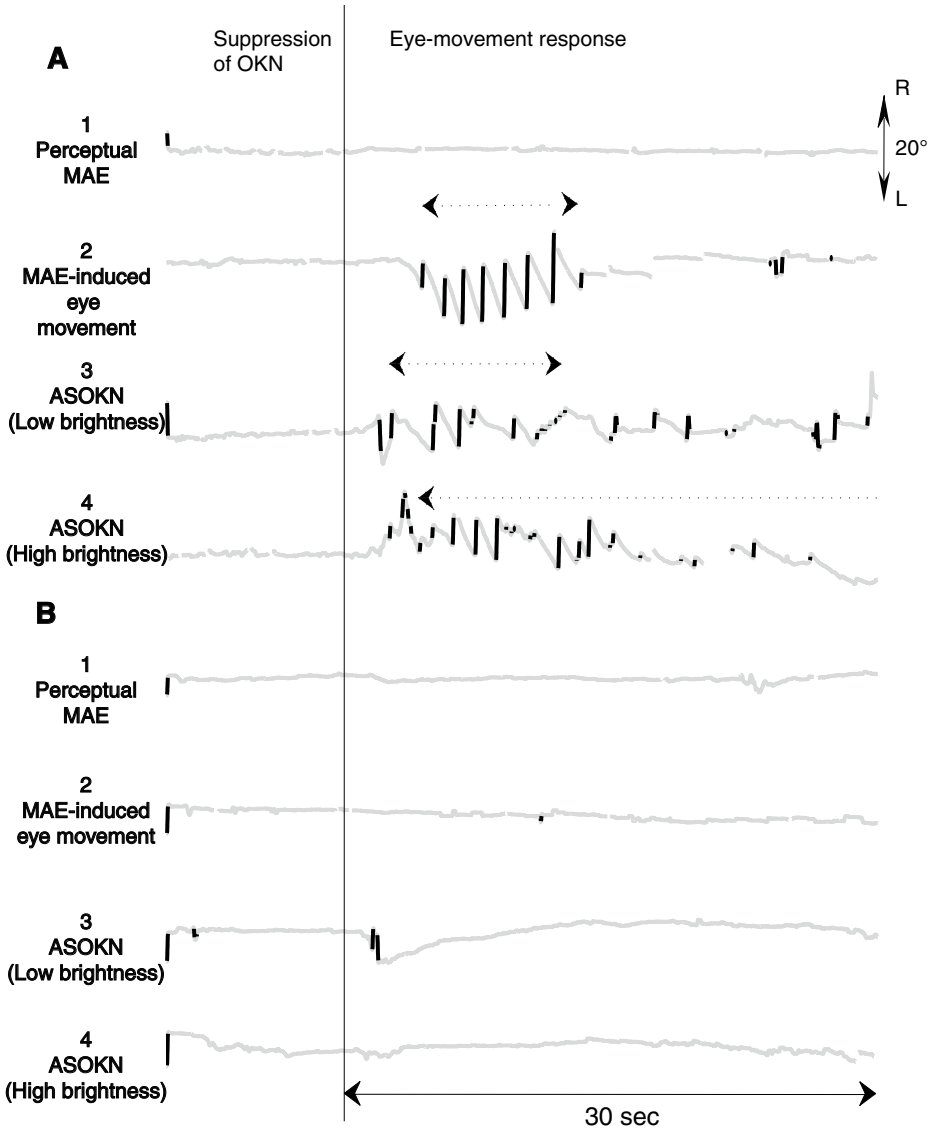


**Fig. 2** Typical examples of eye movements in **a** Subject 1 and **b** Subject 13. Condition number and name are shown on the *left side* of each eye position trace. **a** Subject 1 held gaze stable during the period of suppression of OKN in Conditions 1–4. In Condition 1, gaze stayed stable in the last minute due to the strong fixation stimulus. The subjects reported a perceptual MAE. In Condition 2, the MAE-induced eye movements were found after suppression of OKN. In Conditions 3 and 4, ASOKN was found after suppression of OKN. **b** Subject 13 also held gaze stable during the period of suppression of OKN in Conditions 1–4. In Condition 1, the subject reported a perceptual MAE. In Conditions 2–4, no eye movement was found after suppression of OKN. Slow phases (velocity <30 deg/s) are marked in *gray*, and fast phases are marked in *black*

Afternystagmus after suppression of optokinetic nystagmus (ASOKN)

Whether ASOKN requires the presence of RAI was tested in Conditions 3 (low brightness) and 4 (high brightness). Ten subjects did not generate ASOKN in either condition (see Fig. 2b). Two subjects had ASOKN only in Condition 4, while five subjects had it in both conditions (see Table 1). Duration and maximum slow-phase velocity of ASOKN between the two conditions were compared to determine the influence of stimulus brightness on ASOKN. The duration of ASOKN was significantly longer in the high-brightness condition (paired one-tailed $t$ test, $t = 2.69$, $p = 0.0361$). However, no significant difference was found between the maximum slow-phase velocities of ASOKN in Conditions 3 and 4 (paired one-tailed $t$ test, $t = 1.85$, $p = 0.1125$).

Interestingly, all subjects who had ASOKN in Condition 4 demonstrated MAE-induced eye movements in Condition 2. Two subjects, who showed MAE-induced eye movements in Condition 2 and reported no RAI in Condition 3, did not generate ASOKN in Condition 3 (Table 1).

## Discussion

We investigated the mechanism responsible for ASOKN, in which slow-phase eye movements are in the opposite direction of previous optokinetic stimulation. This ASOKN was only found in a minority of healthy subjects tested. Five out of 17 subjects (29 %) showed ASOKN after presenting the low-brightness stimuli, while seven out of 17 subjects (41 %) showed ASOKN after presenting the high-brightness stimuli. These percentages are less than in a previous

**Table 1** Summary of experimental data

| Subject | 1 | 2 | | | 3 | | | | 4 | | | | 5 | 6 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Perceptual MAE | MAE-induced eye movements | | | Afternystagmus after suppression of OKN | | | | | | | | OKN | |
| | High brightness | High brightness | | | Low brightness | | | | High brightness | | | | Low brightness | High brightness |
| | | Median Velocity (°/s) | Maximum SPV (°/s) | Duration (s) | Retinal Afterimage | Median Velocity (°/s) | Maximum SPV (°/s) | Duration (s) | Retinal Afterimage | Median velocity (°/s) | Maximum SPV (°/s) | Duration (s) | Median velocity (°/s) | Median velocity (°/s) |
| *1* | ✓ | *−2.4* | *10* | *9* | ✓ | *−1.9* | *10* | *10* | ✓ | *−4.6* | *12* | *45* | *23.4* | *24.8* |
| *2* | ✓ | *−2.5* | *15* | *60* | ✓ | *−3* | *15* | *32* | ✓ | *−3.5* | *23* | *42* | *28* | *29* |
| *3* | ✓ | *−0.7* | *7* | *8* | ✓ | *−0.6* | *7* | *4* | ✓ | *−0.9* | *4* | *6* | *6* | *12.6* |
| *4* | ✓ | *−2* | *8* | *50* | ✓ | *−1.8* | *5* | *15* | ✓ | *−2.1* | *9* | *40* | *24* | *22* |
| *5* | ✓ | *−2.3* | *10* | *16* | ✓ | *−3.1* | *8* | *18* | ✓ | *−3.4* | *8* | *18* | *25.6* | *27.4* |
| *6* | ✓ | *−1.6* | *10* | *22* | × | *0.3* | × | × | ✓ | *−0.5* | *4* | *8* | *23.4* | *23.5* |
| *7* | ✓ | *−0.8* | *3* | *11* | × | *−0.4* | × | × | ✓ | *−0.8* | *2* | *10* | *12.7* | *10* |
| 8 | ✓ | −0.4 | × | × | ✓ | 0.3 | × | × | ✓ | 0 | × | × | 27.2 | 29 |
| 9 | ✓ | 0.4 | × | × | ✓ | 0.2 | × | × | ✓ | 0.4 | × | × | 20.3 | 22.9 |
| 10 | ✓ | −0.3 | × | × | × | 0.3 | × | × | ✓ | −0.1 | × | × | 25 | 23 |
| 11 | ✓ | −0.4 | × | × | ✓ | −0.2 | × | × | ✓ | −0.1 | × | × | 19 | 15 |
| 12 | ✓ | −0.1 | × | × | ✓ | −0.2 | × | × | ✓ | −0.3 | × | × | 16 | 21.4 |
| 13 | ✓ | −0.1 | × | × | ✓ | −0.2 | × | × | ✓ | 0 | × | × | 9.7 | 12.3 |
| 14 | ✓ | −0.1 | × | × | ✓ | −0.1 | × | × | ✓ | 0.4 | × | × | 4.2 | 5.7 |
| 15 | ✓ | −0.2 | × | × | × | −0.3 | × | × | × | −0.2 | × | × | 6.5 | 16.3 |
| 16 | ✓ | 0.1 | × | × | × | −0.1 | × | × | × | 0 | × | × | 19.7 | 20.8 |
| 17 | × | −0.3 | × | × | × | −0.4 | × | × | ✓ | −0.3 | × | × | 8.7 | 7 |
| # of presence | 16 | 7 | | | 11 | 5 | | | 15 | 7 | | | 17 | 17 |

Numbers at the top of the table correspond to the condition number. Subjects reporting a perceptual MAE after Condition 1 were marked with "✓". In Conditions 2–4, the median eye velocity of the first 20 s after the period of suppressed OKN was calculated. Subjects reporting a RAI in Conditions 3 and 4 were marked with a "✓". Moreover, maximum slow-phase velocity (SPV) and duration of the MAE-induced eye movements and ASOKN were calculated only in the subjects who had a nystagmus after suppression of OKN. In Conditions 5 and 6, median eye velocity during the 3-min optokinetic stimulation was calculated. The number in the last row indicates the number of subjects who reported or had perceptual MAE, MAE-induced eye movements, RAIs, and ASOKN. The shaded area indicates the subjects who demonstrated the MAE-induced eye movements in Condition 2

✓ = present; × = absent

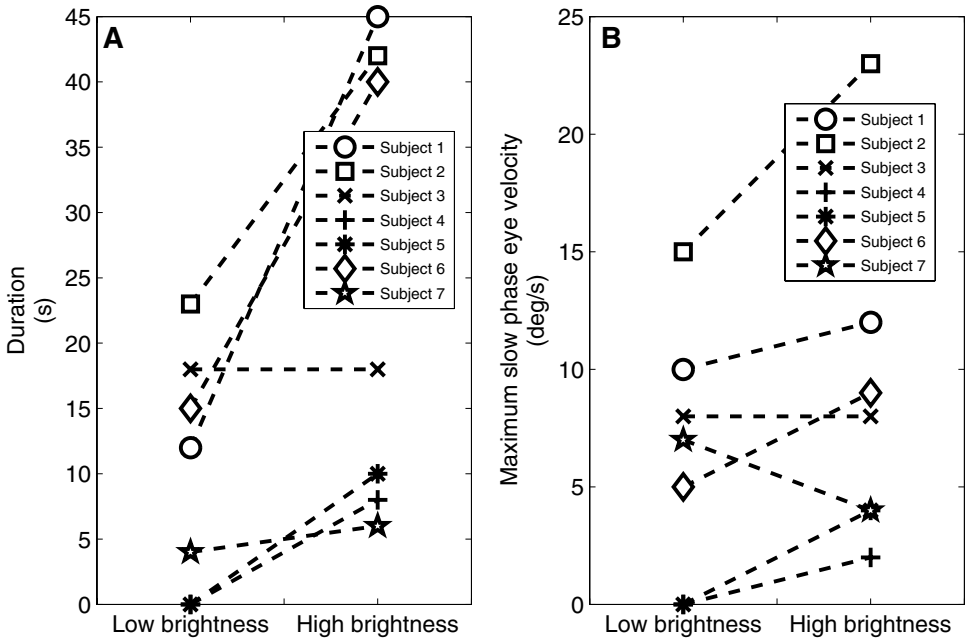


**Fig. 3** Duration (**a**) and maximum slow-phase eye velocity (**b**) of ASOKN in those subjects who had ASOKN in either Conditions 3 or 4

study that reported ASOKN in 14 out of 23 subjects (61 %) (Ventre-Dominey and Luyat 2009, Table 2). In contrast, Fletcher et al. (1990) reported that none of their four subjects (0 %) generated ASOKN in the opposite direction of the preceding optokinetic stimulus. Hence, it seems that ASOKN highly depends on the individual subject and the parameters of the visual stimulation, such as brightness.

We found that ASOKN can be elicited if a subject shows motion-aftereffect-induced (MAE-induced) eye movements and also reports seeing RAIs. Specifically, the subjects who showed MAE-induced eye movements in Condition 2 and saw a RAI in Conditions 3 or 4 generated ASOKN (see Table 1). In the remaining subjects, no ASOKN was recorded. The notion that RAIs are the essential driver of ASOKN was underlined by the fact that the high-brightness stimuli induced a longer duration of ASOKN than the low-brightness stimuli (Fig. 3a), as RAIs persist longer with increasing brightness (Granit et al. 1930; Feinbloom 1938; Nagamata 1951).

The concurrence of RAIs, MAE-induced eye movements, and ASOKN in individual subjects suggests that ASOKN results from an interaction of MAE-induced eye movements and RAIs. The MAE-induced eye movements are very similar to ASOKN with respect to the eye-movement direction and the precondition, i.e. gaze fixation during optokinetic stimulation. But, the MAE-induced eye movements require visual input (Watamaniuk and Heinen 2007), different to the condition of testing ASOKN, where the visual surround is totally dark (Kudo et al. 2002; Ventre-Dominey and Luyat 2009). However, RAIs, if also present after suppression of OKN, can be the visual input for the MAE-induced eye movements. Moreover, the eye-fixed RAIs that move with the MAE-induced eye movements of the eyes may further stimulate eye movements. Such an interaction between the MAE-induced eye movements and RAIs can induce an afternystagmus in the dark after suppression of OKN, i.e. ASOKN. Either RAIs or MAE-induced eye movements alone are not sufficient to generate ASOKN.

### Perceptual motion aftereffect (MAE) and the MAE-induced eye movements

In this study, 16 out of 17 subjects (94 %) reported a perceptual MAE in Condition 1, but only seven out of 17 subjects (41 %) generated MAE-induced eye movements in Condition 2 (see Table 1). Why did not all subjects with a perceptual MAE show MAE-induced eye movements? We speculate that, in these subjects, the perceptual MAE was not strong enough to drive the eyes to follow the motion illusion. Further study is needed.

### Afterimages

We controlled the brightness of the space-fixed yellow dot and the random dot pattern, to induce different strengths of RAIs. Twelve out of 17 subjects (71 %) saw a RAI in Condition 3 (low brightness), while 16 out of 17 subjects (94 %) reported that it appeared in Condition 4 (high brightness). Moreover, 11 out of 12 subjects who saw a RAI in Conditions 3 and 4 reported that the RAI duration was longer and stronger after the high-brightness condition. Our results agree with previous studies, in which brightness of the image positively correlated with the RAI duration

(Granit et al. 1930; Feinbloom 1938; Nagamata 1951; Lu et al. 2006).

### Velocity storage mechanism

Previous studies suggested that ASOKN may be related to some sort of VSM, which is charged by optokinetic signals during the period of suppression of OKN and then causes afternystagmus in the subsequent dark (Kudo et al. 2002; Ventre-Dominey and Luyat 2009). However, Fletcher et al. (1990) suggested that gaze fixation prevents the coupling of optokinetic signals into the VSM. Our study supports the conclusion that it is not the VSM being charged by retinal slip during fixation of a stationary target. In our experimental data, Conditions 5 and 6, which used the same moving random dot pattern, except for the brightness, evoked similar degrees of OKN (see Table 1). In other words, although the brightness was different, the two optokinetic stimuli caused a similar amount of retinal slip. In Conditions 3 and 4, however, we found that the high- and low-brightness conditions significantly influenced the duration of ASOKN (see Fig. 3A); high brightness evoked a longer ASOKN. This phenomenon cannot be well explained by a hypothesis involving a VSM.

### Summary

In summary, we found that ASOKN occurred only if subjects had the MAE-induced eye movements and saw a RAI after suppression of OKN. Our results therefore suggest that ASOKN may be the result of an interaction of MAE-induced eye movements and RAIs.

**Acknowledgments** The authors like to thank Urs Scheifele and Marco Penner for excellent technical assistance. This work was supported by the Swiss National Science Foundation (SNF) grants PMPDP3_139754 (Marie Heim-Vögtlin programme) and 31003A-118069, Zurich Center for Integrative Human Physiology (ZIHP), and Betty and David Koetser Foundation for Brain Research.

## References

Anstis S, Verstraten FA, Mather G (1998) The motion aftereffect. Trends Cogn Sci 2:111–117

Brainard DH (1997) The Psychophysics Toolbox. Spat Vis 10:433–436

Brown JL (1965) Afterimages. In: Graham CH (ed) Vision and visual perception. Wiley, New York, pp 479–503

Cohen B, Matsuo V, Raphan T (1977) Quantitative analysis of the velocity characteristics of optokinetic nystagmus and optokinetic after-nystagmus. J Physiol 270:321–344

Feinbloom W (1938) A quantitative study of the visual after-image. Archives of Psychology (Columbia University) 233

Fletcher WA, Hain TC, Zee DS (1990) Optokinetic nystagmus and afternystagmus in human being: relationship to nonlinear processing of information about retinal slip. Exp Brain Res 81:46–52

Granit R, Hohenthal T, Uoti A (1930) On the latency of negative afterimages in relation to brightness of stimulus. Acta Ophthalmol 8:137–154

Kleiner M, Brainard D, Pelli D (2007) What's new in Psychtoolbox-3? Perception 36 ECVP Abstract Supplement

Korenaga K, Makishima K, Yosida M, Kudou K, Higuchi S (1996) Experimental study on directional asymmetry of vertical OKN and OKAN. J Otolaryngol Jpn 99:1085–1094

Kudo K, Yoshida M, Makishima K (2002) Reverse optokinetic afternystagmus generated by gaze fixation during optokinetic stimulation. Acta Otolaryngol 122:37–42

Laurens J, Angelaki DE (2011) The functional significance of velocity storage and its dependence on gravity. Exp Brain Res 210:407–422

Lu H, Zavagno D, Liu Z (2006) The glare effect does not give rise to a longer-lasting afterimage. Perception 35:701–707

Mather G, Verstraten FA, Anstis S (1998) The motion after-effect. MIT Press, Cambridge

Nagamata H (1951) Contribution to the knowledge of after-images. Acta Soc Ophthalmol Jpn 55:802–806

Ohm J (1921) ber optischen Drehnystagmus. Klin Monbl Augenheilkd 68:234–235

Pelli DG (1997) The VideoToolbox software for visual psychophysics: transforming numbers into movies. Spat Vis 10:436–442

Raphan T, Cohen B, Matsuo V (1977) A velocity-storage mechanism responsible for optokinetic nystagmus (OKN), optokinetic after-nystagmus (OKAN) and vestibular nystagmus. In: Baker R, Berthoz A (eds) Control of gaze by brain stem neurons, developments in neuroscience, vol I. Elsevier/North-Holland Biomedical Press, Amsterdam

Raphan T, Matsuo V, Cohen B (1979) Velocity storage in the vestibuloocular reflex arc (VOR). Exp Brain Res 35:229–248

Shimojo S, Kamitani Y, Nishida S (2001) Afterimage of perceptually filled-in surface. Science 293:1677–1680

Spigel IM (1960) The effect of differential post-exposure illumination on the decay of the movement aftereffect. J Psychol 50:209–210

Thompson P, Wright J (1994) The role of intervening patterns in the storage of the movement aftereffect. Perception 23:1233–1240

Ventre-Dominey J, Luyat M (2009) Asymmetry of visuo-vestibular mechanisms contributes to reversal of optokinetic after-nystagmus. Exp Brain Res 193:55–67

Verstraten FAJ, Fredericksen RE, Grusser OJ, Van de Grind WA (1994) Recovery from motion adaptation is delayed by successively presented orthogonal motion. Vision Res 34:1149–1155

Watamaniuk SN, Heinen SJ (2007) Storage of an oculomotor motion aftereffect. Vision Res 47:466

Wohlgemuth A (1911) On the aftereffect of seen movement. Br J Psychol 1(Suppl):1–117